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Activation of DNA damage checkpoint pathways during skeletal myoblast differentiation and apoptosis

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Abstract

A subset of skeletal myoblasts undergo apoptosis rather than differentiation when cultured in differentiation media (DM: absence of growth factors). While the muscle regulatory transcription factor MyoD is known to control the process of differentiation, our lab has recently discovered that MyoD is also controlling the apoptotic process in response to culture in DM by direct up-regulation of the pro-apoptotic Bcl2 family member PUMA. We similarly discovered that MyoD plays a role in the increased expression of PUMA and apoptosis in response to the DNA damaging agent, etoposide. This led to the hypothesis that culture in DM may lead to stalled replication forks during DNA synthesis that are “recognized” as DNA damage. We are testing our hypothesis by determining if culture in DM results in the activation of pathways known to respond to DNA damage. We have determined that p38, p53, and c-abl are all up-regulated in response to culture in DM. Next, we will determine the significance of MyoD to the increased expression of these molecules.

**McNair Scholar*